A DISSERTATION ON

HYPOPYON UVEITIS

M.S. DEGREE BRANCH (III)

OPHTHALMOLOGY

THE TAMILNADU

DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI, TAMILNADU

FEBRUARY 2006

CERTIFICATE

This is to certify that this dissertation entitled "HYPOPYON UVEITIS" submitted by DR.N. PARVATHA SUNDARI to the faculty of Ophthalmology, The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the requirement of the award of M.S.Degree Branch III (Ophthalmology) is a bonafide reserach work carried out by her under our direct supervision and guidance.

DR. R. Geetha Ramani, M.S. D.O.,

PROFESSOR & H.O.D.
Department of Ophthalmology,
GOVT. RAJAJI HOSPITAL &
MADURAI MEDICAL COLLEGE
MADURAI.

DECLARATION

I, Dr. N. Parvatha Sundari solemnly declare that the dissertation titled "HYPOPYON UVEITIS" has been prepared by me.

This is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfilment of the requirement for the award of M.S., degree (Branch III Ophthalmology) Examination to be held in FEBRUARY 2006.

Place : Madurai

Date : Dr. N. PARVATHA SUNDARI

ACKNOWLEDGEMENT

I am grateful to The Dean, Madurai Medical College, Madurai for permitting me to do the study.

I am extremely grateful to Professor Dr. R. GeethaRamani. M.S. D.O., Professor and HOD of Ophthalmology, Madurai Medical College, Madurai for the able guidance, inspiration and encouragement she rendered at every stage of the study.

I take this opportunity to express my deep sense of gratitude to Professor Dr. R. Unnamalai M.S. D.O. for her guidance and help for executing my study.

I am grateful to Dr. K. Sivakumar, Asst. Professfor, Department of Ophthalmology for his valuable guidance, support and encouragement rendered to me during the study.

I am extremely grateful to all the Assistant professors, Department of Ophthalmology for having helped during the study.

I am extremely grateful to all the Assistant professors of Medicine and Rheumatology department for their support during the study.

I thank my study subjects who formed the back bone of the study and without whom this work would not have been possible.

Last but not the least, I thank “God, the Almighty” for being my guiding light all the way.

CONTENTS

INTRODUCTION

1. Hypopyon :

Hypopyon is a sterile collection of inflammatory cells in the inferior angle of the anterior chamber.

2. Historical Background :

Hypopyon was first considered to lie in the corneal substance and represented the gravitation of pus between the lamellae. It was called “onyx” so called from its resemblance to the root of a finger nail, an appearance which is now called hypopyon (under, pus). It was thus for a long time thought to be derived from the cornea, but the sterility of the material and the presence of pigmented granules in leucocytes demonstrated its origin from iris and ciliary body.

3. Hypopyon Uveitis

Hypopyon uveitis refers to certain specific uveitic entities that are characterized by a sterile collection of leucocytes & variable amounts of fibrin indicating a severe inflammatory process.

4. Magnitude of the problem :

The prevalence of uveitic entities that are likely to present with Hypopyon are in order of their prevalence. Idiopathic uveitis, Anky losing – spondylitis uveitis, other HLA B 27, uveitis (Reiter's syndrome, Inflammatory bowel disease), Behcet's disease, Herpes simplex keratouveitis, toxoplasmosis, toxocariasis, Histoplasmosis, ocular candidiasis, ocular syphilis, Traumatic iridocyclitis, leukemia, lymphoma (Maquerade syndrome)

ANATOMY

Uvea Includes The Iris, Ciliary Body and Choroid :

IRIS

The Iris is a diaphragm whose periphery is attached to the anteromedial aspect of the ciliary muscle. It forms the posterior boundary of the anterior chamber, is about 12mm in diameter, 0.6 mm thick at the collarette (its thickest point) and 0.5 mm at its periphery. It is perforated by the pupil, which is displaced slightly nasal to centre, and its papillary margin rests posteriorly on the anterior surface of the lens.

In cross-section the iris shows a stromal layer anteriorly and a double epithelial layer posteriorly. The stroma is continuous with that in the ciliary body at the iridial periphery.

Layers of the Iris :

The layers of the iris are :

1. the anterior border layer, a network of fibroblasts and pigment cells. The degree of pigmentation determines iridial colour.
2. the anterior leaf of stroma
3. the posterior leaf, contains nerves and blood vessels.
4. the sphincter muscle (1mm wide) in the papillary zone, readily visible in blue irides. Contraction, induced by parasympathetic stimulation, causes pupil constriction
5. the dilator muscle, chiefly in the ciliary zone, consists of processes of the anterior myoepithelium extending as a sheet central to the sphincter's margin. Contraction, induced by sympathetic innervation, causes pupil dilation
6. the anterior myoepithelium, pigmented
7. the posterior pigmented epithelium
8. a basal lamina

The major arterial circle of the iris, located in the anterior face of the ciliary body, gives off numerous radial branches supplying iridial tissues. It is reinforced by an inconstant minor arterial circle within the collarette.

CILIARY BODY :

The ciliary body extends from the scleral spur to the ora serrata, a scalloped anterior margin of the retina. In meridional section it is divided into a posterior pars plana (3.5-4.5mm) wide) and an anterior pars plicata (about 2mm wide), which bears 70-80 mm radial ciliary processes and encloses most of the ciliary muscle.

The width of the ciliary body is 4.5-5.2 mm nasally, 5.6-6.3 mm temporally .

Layers of the ciliary body :

From within outwards these are :

1. an internal limiting membrane
2. a non-pigmented epithelium which in the region of the ciliary processes, is specialized for secretion of aqueous humour, which is under autonomic and humoral control;
3. a pigmented epithelium

4. a stroma of connective tissue containing vessels and nerves and non-striated muscle; the meridonal part of the ciliary muscle is chiefly in the pars plana, its radial and circular parts in the pars ciliaris.

A supraciliary zone, filled with pigmented cells and loose connective tissue, lies outside the ciliary body.

AQUEOUS HUMOUR :

This clear fluid is secreted at a rate of about 2.5ul/min and drains from the anterior chamber chiefly by bulk flow through the conventional drainage pathway, but also via a uveoscleral route from the anterior chamber into the supraciliary space. Secretion is under autonomic and humoral control. The rate of secretion and drainage maintains the intraocular pressure within the narrow range of 10-21 mmHg.

The aqueous humour supplies nutrients to the cornea and lens and exchanges gases and metabolities with these avascular tissues, serving the role of a vascular supply.

CHOROID :

The choroid is chiefly a vascular layer nutritive to outer retinal layers. Its anterior boundary is the ora serrata and posteriorly it ends around the optic nerve head. It is supplied chiefly by the short posterior ciliary arteries, but also by the long posterior vessels, which traverse it in the nasal and temporal horizontal meridians.

Venous blood drains by multiple tributaries into several venae vorticosae.

Layers of the choroids :

The layers of the choroid coat are :

1. an external layer of large vessels (Haller's layer)
2. a middle layer of vessels intermediate in size (Sattler's layer)
3. an internal layer of fenestrated capillaries, the choriocapillaris, intimately related to Bruch's membrane

   All these vessels lie in a loose matrix of connective tissue rich in melanocytes, and permeated by nerve fibres.

4. Bruch's membrane , about 2 um thick, is formed by the basal laminae of the choriocapillaris and retinal pigment epithelium with an intervening zone of collagen and elastic tissue. It provides a barrier restricting movement of large molecules from the choroids. It is about 2 um thick, rich in collagen, but also contains a middle elastic zone.

ANTERIOR CHAMBER :

The anterior chamber is a compartment filled with aqueous humour, lying behind the cornea and in front of the iris and the lens in the pupillary space. The axial depth of the chamber is about 3.0mm (range 2.6-4.4mm) and its volume in the emmetrope is about 250 µl. (The posterior chamber's volume is about 60 µl.

The angle of the chamber is its most peripheral part and gives access to the drainage structures by which aqueous leaves of the eye (hence the term drainage angle).

BLOOD AQUEOUS BARRIER

- This includes all the barrier to movement of substances from the plasma to aqueous tumor.
- In ciliary body- Vascular endothelium

  Basement membrane, stroma

  Pigment and non pigment epithelium - tight junction of NPE which connects the apical portion of adjacent NPE cells.
- Allows passage of some ions & water
- Responsible for maintaining the chemical composition of aqueous
- Breakdown of BAB - ↑ aqueous protein concentration
  - ↑ WBCS
  - ↑ IOP followed by decreased IOP
- brings mediators of cellular humoral immunity to interior of eye.
- responsible for development of cataract and synechiae formation
- BAB breaks – protein rich fluid collect in cysts beneath & in between epithelial cells of ciliary body – then burst into posterior chamber leading to increased protein concentration.

Stimuli that Breakdown blood aqueous barrier.

a) Trauma : Injury of iris or lens – contusion, paracentesis

b) Chemical irritant : acid, alkali, formaldehyde

c) Neural activity : stimulation of V . Nerve

d) Immunogenic activity

e) Endogenous mediators

- Histamine, Bradykinin
- PG, serotonin
- Acetylcholine
- Miscellaneous

f) Bacterial endotoxins

g) X radiation

h) Infrared rays

i) Laser energy

PATHOGENESIS & IMMUNOLOGY OF HYPOPYON UVEITIS

Inflammation of the iris is characterised by the dilatation of blood vessels of iris, impairment of capillary wall, exudation of protein rich fluid and leucocytosis as in any inflammation.

a) Peculiarities of the iris :

The iris is composed of a stroma containing connective tissue cell, usually pigmented but largely unpigmented in blue irides, the posterior surface is covered by two layers of pigmented epithelium, the anterior surface is covered with a single layer of endothelium except at region of the crypts at ciliary border. The bulk of stroma is formed by blood vessels. The majority of the vessels follow a course radial with the centre of pupil. Additional vessels pursue a concentric course in a corkscrew fashion around the pupil. The diameter of the capillaries here is relatively large, their endothelium is nonfenesterated surrounded by a basement membrane, associated pericytes and a zone of collagenous filaments. In the region of collarete there is an anastomosis between the arterial and venous arcades, the “major arterial circle” is however located in the ciliary

body. The iris stroma itself is typically loosely formed, composed of pigmented and non pigmented cells, collagen fibrils and a matrix of hyaluronidase sensitive mucopolysaccharide. The tissue spaces in the stroma communicate directly with the AC through crypts, allowing easy transference of fluid between the iris and anterior chamber. Because of this extreme vascularity of iris, the peculiar distribution of the vessels and the looseness of stroma, there is scope for an unusually large amount of exudation on one hand and swelling on the other end.

PATHOGENESIS :

The first sign of inflammation is the ‘vascular reaction”. There is hyperemia, massive dilation of the smaller vessels engorgement with RBCs and PMN cells. The PMN cells escape from the vessels wall in large quantity, surrounding the vessels as a cuff, the lumen is blocked with fibrin, pus cells and organism if any, causing further engorgement of the vessels. The surrounding stroma is packed with exudative cells, erythrocytes PMNS, Lymphocytes etc), the latter more in exogenous inflammations.

The stromal cells are swollen, the processes disappear, becomes round, the nuclei, stain poorly, and they begin to necrose or wander out and mix with the surrounding exudative cells. The chromatophores also show similar disintegration changes, they break up, their pigment granules scattered and lying loose in tissue or engulfed by leucocytes.

In iris, the whole structure is uniformly thickened and necrotic and from both aspects there pours out a fibrinous exudates rich in leucocytes which escapes into the AC making the aqueous cloudy and rapidly forming a hypopyon the consistency of which depends on the proportion of fibrin which encloses the leucocytes it meshes.

As a rule the hypopyon is white or yellow in colour and fluid in consistency, but if the proportion of fibrin is high it may be grey, translucent and semisolid. With fibrin contraction this hypopyon may shrink and disappear.

CLASSIFICATION OF HYPOPYON UVEITIS :

I (a) Hypopyon uveitis caused by corneal infection :

Bacterial keratitis	-	Streptococcus pneumoniae
		Neisseria gonorrhea
		Proteus
		Pseudomonas
		E – coli
Viral keratitis	-	Herpes simplex keratitis
		Herpes zoster keratitis
		Measles
Fungal keratitis	-	Candida albicans
		Fusarium

b) Hypopyon caused by Intraocular inflammation

Non infections causes	-	Behcet's
		Severe hyperacute Iridocyclitis
		HLA B 27 uveitis
		Herpes simplex ketarouveitis
		Herpes zoster ketarouveitis
		Intraocular tumors (Melanoma, RB)

Retained Intraocular foreign body

Lens induced uveitis.

Infectious causes : Toxocara endophthalmitis

C) Non Inflammatory pseudohypopyon :

Intraocular tumors - Retinoblastoma

Malignant Melanoma

Reticulum cell sarcoma

Leukemia, Lymphoma

Synchisis syntillans

II - Classification based on the anatomical site :

i) Anterior uveitis with hypopyon

Corneal infection

HLA B 27 uveitis

Behcet's

Herpes simplex keratouvetis

Herpes zoster keratouveitis

Traumatic Iridocyclitis

Lens induced uveitis

Retained IOFB

ii) Posterior uveitis with hypopyon

Behcets

Toxoplasma Retinochoroiditis

Toxocera endophthalmitis

Necrosis secondary to I.O. tumours

Retained IOFB

Endogenous endophthalmitis

Masquerade syndrome

iii) Pan uveitis with hypopyon

Behcet's

Masquerade syndrome

Leptospirosis

Endogenous endophthalmitis

III) Classification based on laterality :

Unilateral : Corneal infection - lens induced uveitis

Herpes simplex keratouveitis - leptospirosis

Herpes zoster keratouvieitis - HLA B27 uveitis

Masquerade syndrome - Behcet's

Retained I O F B

Toxoplasmosis

Toxocariasis

Endogenous endophthalmitis

Bilateral : Behcet's. HLA B 27, toxoplasmosis, leptopirosis

IV) Sex predilection :

Male : Behcet's

HLA B 27 uveitis

Male = Female :

Secondary to corneal infection

Herpes simplex viral keratouveitis

Herpes zoster viral keratouveitis

Masquerade syndrome

Retained I O F B

Toxoplasma Retinochoroiditis

Toxocariasis

Endogenous endophthalmitis

Lens induced uveitis

Leptospirosis

Severe acute iridocyclitis.

V) Presentation :

Acute uveitis with hypopyon

secondary to corneal infection

HLA B 27 uveitis

Behcet's

Any severe iridocyclitis

Herpes simple keratouveitis

Herpes zoster keratouveitis

Retained IOFB

Toxoplasmosis

Toxocariasis

Endogenous endophthalmitis

Lens induced uveitis

Leptospirosis

Chronic - Behcet's

Masquerade syndromes

Toxocariasis

Retained IOFB

VI Pathology :

Non – granulomatous : HLA B 27 uveitis

Behcets

Leptospirosis

Granulomatous : Viral

Lens induced uveitis

Masquerade syndromes

Toxoplasmosis

Toxocariasis

Endogenous endophthalmitis

Leptospirosis

Non granulomatous or Granulomatous :

Behcet's

Leptospirosis

VII Based on age pattern :

In children : Toxocara

Masquerade - Retinoblastoma

Leukemia

Endogenous endophthalmitis

In Adolescents

- toxocara endophthalmitis
- viral uveitis
- masquerade syndromes
- endogenous endophthalmitis

In Adults

- HLA B 27 uveitis
- Behcet's
- Viral uveitis
- Toxoplasma retinochoroiditis
- Toxocara endophthalmitis
- Leptospiral uveitis

In old age :

- Behcet's
- Lens induced uveitis
- Leptospirosis
- Masquerade syndromes

VIII - Based on gross nature of hypopyon

White - Behcets

Yellow	-	HLA B 27 uveitis
		Lens induced uveitis
		Leptospirosis
		Toxoplasmosis
		Toxocariasis
		Endogenous endophthalmitis
Blood tinged	-	Viral keratouvetis
		Leukemia
		Juvenile xanthogranuloma
Pink hypopyon	:	Serratia endopthalmitis
Brown hypopyon	:	Malignant melanoma
Black hypopyon	:	Listeria monocytogenes
		Malignant melanoma
Inverse hypopyon	:	Silicone oil

IX - Rare causes of hypopyon uveitis :

a) TB associated hypopyon

b) Rifabutin therapy

c) Following laser procedures

d) Leprosy

CLINICAL FEATURES OF HYPOPYON UVEITIS

I - SYMPTOMS:

1) Pain :

Pain is due to ciliary spasm, ciliary body is innervated by fifth nerve. This pain radiates to the whole region distributed by this nerve including periorbital region and eye itself . Inflammation can occur in the ciliary body or distal to the ciliary body. inflammation in the cornea can cause a retrograde axon reflex vasodilation & swelling in the ciliary body.

2) Photophobia :

Pain is caused by exposure to light. It occurs in iritis & irdocyclitis. Axon reflex is mainly responsible for ciliary spasm . Also normal pupillary stimulation to light tends to cause iris and ciliary body movement which further irritates nerves.

3) Lacrimation & tearing :

is due to fifth nerve irritation .

4) Blurred vision & opacities

is caused by cloudy medium , such as from cells & vitreous bands. Floaters and spots in front of eye are commonly caused by cells and precipitates in the deep vitreous. Macular edema which frequently occur with anterior and posterior uveitis can produce blurred vision.

II Signs:

a) Ciliary injection is due to

- ring of dilated episcleral vessels radiating around the limbus.
- Topical phenylephrine does not cause blanching

b) Cornea :

Keratic precipitates:

Aggregates of inflammatory cells deposited on the back of cornea from the aqueous humour . Altered endotheluim may attract these cells in a linear vertical formation (Turk's line) or in a triangular formation with apex above (Arlt's Δ), Their distribution results from the convection current in the AC that rises along the warm iris & falls along the cool cornea. Clinically Kps may be small

,medium ,large & coalescent they consist of Polymorphomuclear cells, lymphocytes , plasma cells & macrophages.

Non - granulomatous type – fine to medium size Kps consist of lymphocytes , plasma cells & pigment. Graunulomatous type – Kp's are large , greesy , muttonfat appearance upto 1mm in diameter consists of epithelioid cells and mononuclear macrophages. Fresh Kps – white , round ,hydrated. Old Kps – Pigmented , shrunken, crenated edges, glassy appearance.

Other corneal findings:

Corneal dendrites, Interstitial keratitis, Band keratopathy, Vascularisation, Corneal edema, Corneal guttata

Anterior chamber:

i) Flare: proteinacious exudate or transudate from iris or CB due to inflammation . Normally aqueous humor appears optically empty to passage of light. The visibility of beam of light implies presence of protein.

Grading of flare : light intensity is turned to maximum and light focused at an angle to plane of iris of size 2mm x 1mm slit .

faint – just detectable - +1

moderate – iris details clear - +2

marked – iris details hazy - +3

intense with severe fibrinous exudate - +4

ii) Cells : Usually indicates active inflammation in the ciliary body. They are primarily lymphocytes , macrophages & monocytes , PMNS may be present early in the course of disease.

Grading : Slit 2*1mm is set at an oblique angle and focussed just behind the cornea & cells are accurately counted. chronic flare in the absence of cells is not a sign of inflammation, Since the flare is due to permanent breakdown of blood aqueous barrier.

Graded as follows:

5 – 10 cells - +1

11 – 20 cells - +2

21 -50 cells - +3

>50 cells - +4

Hypopyon : is collection of leuocytes that settle in the lower angle of the anterior chamber. It is related to the no of cells in AC. It is related to the presence of sufficient fibrin to cause the cells to clump & settle.

Iris:

a) **Synechiae:** adhesions between the iris & lens capsule or between iris & cornea near the AC angle . Inflammation is accompanied by release of mediators that promote fibrin deposition , clotting & fibroblast proliferation that causes synechiae. Presence of synechiae indicates that the inflammation has been chronic or recurrent. Most commonly synechiae occurs at pupillary border. With severe inflammation adhesion of entire posterior iris surface to the anterior lens capsule occurs.

b) Iris Nodules :

Accumulation of inflammatory cells in the iris or on its anterior surface. This may be grey or white & translucent & often covered with pigment. Two types are Koeppe & bussacca nodules.

Other iris findings:

-tiny shiny spots on surface of iris in chronic iritis – russel body

-prominent iris vessels that follow normal anatomy

-neovascular rubeotic vessels

Pupils:

Miosis :

Iris irritation causes a release of prostaglandins & miosis , due to axonal reflexes caused by irritation of the fifth cranial nerve endings else where in eye can lead to antidromic iris vasodilation & subsequent papillary constriction

- Dilation of the radially disposed vessels causes mechanical miosis.

a. Sluggish reaction

b. Pupillary membrane

Fibrovascular membrane develops on the anterior surface of lens across the pupillary margin.

Lens:

a. Chronic & recurrent acute iridocyclitis leads to posterior subcapsular cataract

b. Steroid use also contributes to cataract formation.

c. Fibrous tissue proliferation may lead to epilenticular membrane formation, lysophosphatidylcholine & macrophages contributes to the formation of cataract.

d. Cyclitic membrane:

Fibrous proliferation behind the lens.

Vitreous:

-Characterised by increased cells & protein which arises from ciliary body, choroid, retina. These lead to formation of snowball opacities, Exudates over pars plana, Vitreous strands

Cyclitic membrane, Ciliary body detachment, Hypotony.

Fundus Examination:

Posterior segment findings include

-Cystoid macular edema, Epiretinal membrane, SRNVM, Optic neuritis, Vasculitis, Choroiditis, Vascular sheathing, Retinal haemorrhage, cottonwool spots.

Optic nerve:

The changes in the optic Nerve head are

Disc hyperaemia, Papillitis, Glaucomatous damage, Optic atrophy, Intraocular pressure:

a. Low:

-Ciliary shock

-Decreased aqueous production

-Increased alternative outflow

b. High

-Meshwork clogged by inflammatory cells or debris

- Trabeculitis

-Pupillary block

-Peripheral anterior synechiae & angle closure

DISCUSSION OF SPECIFIC UVEITIC ENTITIES WITH HYPOPYON & REVIEW OF LITERATURE

Common Causes of Hypopyon uveitis :

1. HLA-B27 uveitis
2. Endophthalmitis
3. Leptospiral uveitis
4. Behcets disease
5. Viral keratouveitis
6. Lens induced uveitis
7. TB associated uveitis
8. Post operative sterile endopthalmitis
9. Idiopathic uveitis

1) HLA-B27 uveitis

It denotes a genotype located on the short arm of chromosome 6.50 to 60 % of patients with acute iritis may be HLA-B27 positive. Seronegative spondylarthropathies are strongly associated with both acute anterior uveitis & a positive HLA-B27. They do not have a positive rheumatoid factor.

It is the commonest cause of endogenous hypopyon formation with 12-14.5% of HLA B27 patients having hypopyon uveitis. Posterior segment manifestation of HLA B 27 uveitis include-Severe vitreous inflammation, papillitis, retinal vasulitis, pars plana enudates (31). Kearney et al (21) observed an incidence of hypopyon uveitis in 12% and D'allexenandro (7) observed an incidence of hypopyon uveitis in 14.5% of HLA B 27 patients.

The Seronegative spondylarthropathies are

Ankylosing spondylitis, Reiter Syndrome , Inflammatory Bowel disease, Psoriatic arthritis, Post infectious or reactive arthritis.

ANKYLOSING SPONDYLITIS :

This primarily involves the sacro-iliac joins & axial skeleton .

Clinical features:

Usually presents with sacroilitis or iritis

- Acute iritis is recurrent & nongranulomatous in 30% both eyes are affected ,in severe cases fibrinous aqueous exudates occur .

-Other features are aortitis , aoritic regurgitation,

pulmonary apical fibrosis

REITER SYNDROME :

It is defined as an episode of peripheral arthritis of more than one months duration occurring in association with urethritis or cervicitis. About 70% are positive for HLA-B27.

CLINICAL FEATURES:

1. Nonspecific urethritis
2. Polyarthritis
3. Conjunctivitis or Iritis

- occurs in young adult males may be triggered by episodes of diarrhea or dysentry without urethritis
- Arthritis begins within 30 days of infection – knee & ankles are affected
- Iritis –acute nongranulomatus iritis occurs Conjuntivitis may be mucopurulent & papillary
- Keratitis is punctuate & subepethelial

MAJOR DIAGNOSTIC CRITERIA :

a. Keratoderma blenorrhagicum – a scaly erythematous disorder of palms & soles .
b. Circinate balanitis – a persistent scaly erythematous circumferential rash of the distal penis.

MINOR DIAGNOSTIC CRITERIA:

Planter fasciitis, achillistendinitis, sacro ileitis , nail bed pitting, palatal ulcers , tongue ulcers.

INFLAMMATORY BOWEL DISEASE

Ulcerative colitis & crohn disease are associated with acute iritis.

20% have sacroiliitis & 60% are HLA-B27 Positive.

PSORIATIC ARTHRITIS:

It is charaterised by arthritis involving the distal IPjoints along with nail pitting & ocular features like conjunctivitis acute iritis , keratitis. (13)

BECHET 'S DISEASE

It is a generalized occlusive vasculitis of unknown cause. It affects young males,

Classic triad is

-Acute iritis with hypopyon,

-Apthous stomatitis,

-Genital ulceration,

OCULAR FEATURES :

Hypopyon uveitis is a feature in nearly 19-30% of cases. The hypopyon is transient and is dramatic & recurrent. (20)

Other features are retinal vasculitis, retinal haemorrhages, macular edema, focal areas of retinal necrosis, ischemic AION, vitritis.

GENERAL FEATURES :

a) mucous membrane lesion are oro genital ulcerations
b) skin lesion –erythema nodosum, acneiform lesions, cutaneous hypersensitivity, thrombophlebitis
c) vascular lesions –obliterative thrombophlebitis, arterial occlusion
d) other features – arthropathy,GI lesion and CNS involvement

III.VIRAL KERATOUVEITIS:

Varicella Zoster is a DNA virus that commonly infects human. It is capable of establishing latency after primary infection and disease reactivation due to latent virus may occur. Chicken pox is the primary human infection with varicella.

Herpes Zoster ophthalmicus is frequently associated with an acute mild non granulomatous bilateral iritis or iridocyclitis.(30)

- cutaneous vesicles at the side of tip of nose (Hutchison sign)indicate fifth cranial nerve involvement and greater likelihood that eye will be affected.
- the stellate KPs assume a diffuse distribution as opposed to the usual distribution in the inferior 1/3 of cornea.
  - KP are fine and fibrillar and stellate pattern.

Other associated features→ Dentritic ulcer, Glaucoma, ↓corneal sensation, Hyphaema, Segmental iris atrophy, Viral retinitis, Retinal artery occlusion, Scleritis

IV.ENDOPHTHALMITIS:

Refers to intraocular inflammation predominantly involving the vitreous cavity and anterior chamber of the eye. Contiguous ocular structure such as retina or the choroids may also be involved.

There are two types

- Non infectious
- Infectious

The most common sign of endophthalmitis are

a. Pain, ↓vision, Hypopyon, Vitritis, Conjuctival hyperemia, chemosis,eyelid edema, corneal edema.

Infectious endophthalmitis :

Classified according to the circumstances by which the infecting organism is introduced into the eye.

a)Exogenous endophthalmitis ; this accounts for most cases

I. Postoperative endophthalmitis –through a surgical incision
II. Post traumatic endophthalmitis – through a traumatic laeration
III. Bleb associated endophthalmitis-a conjuntival filtering bleb

b) Endogenous endophthalmitis – gains access to the eye from the internal environment or haematogenously.

A) Post operative endophthalmitis :

Eyelid &conjunctiva are the primary source of infection.The organism may be the normal ocular surface flora such as staphylococcus species and propionibacterium acne.

Other sources are Lacrimal system, Blepharitis, Contaminated eye drops, Contaminated surgical instruments ,IOL or irrigation fluids, major breach in sterile technique.

a) acute postoperative endophthalmitis

i) mild – most common organism is staphylococcus epidermidis

(1) sterile: onset is within fourteen days. symptoms are photophobia, floaters. They have a slow progression , vision >20/400 , ±hypopyon ,mild vitritis fundus is visible.

ii) severe : most common organism is staphylococcus aureus, streptococcus species, gram negative bacteria. Onset is in fourteen days. Symptoms are pain, decreased vision. They have a rapid progression , vision <20/400 , ±hypopyon, marked vitritis, fundus not visible

b) Chronic post operative endophthalmitis :

Most common organism : propionibacterium acne, staphylococcus epidermidis, fungus

Onset : two weeks to two years

Symptoms : photophobia, hazy vision

Here hypopyon is less common staphylococcus epidermidis infection presents with non granulomatous inflammation. Fungal endophthalmitis begins 3 months after surgery (40) and is most commonly caused by candida species. P. acne endophthalmitis develops 2 months to 2 years following cataract surgery and is characterized by granulomatous KP , a small hypopyon , vitritis , and a white plaque containing Propionibactrium acne and residual lens material sequestered within the capsular leaf . Rarely caused by YAG capsulotomy which allows the dissemination of sequestered pathogen from the capsular leaf into the vitreous cavity and anterior chamber.

c) Associated with filtering bleb ;

Organism : Streptococcus Species Haemophilus influenzae

Onset : anytime Symptom; Red eye , discharge , pain ,decreasing vision

Clinical features : Infected bleb , hypopyon , vitritis

d) Endogenous endophthalmitis :

Results from the blood borne spread of bacteria or fungi during generalized septicemia.

Source: a remote non ocular source like an injected IV line or an injected organ as in endocarditis, GIT disorders,pyelonephritis, meningitis or osteomyelitis. Diabetics, immunosuppressed are predisposed. I.V drug abusers, indwelling catheters ,immediate or intermediate postpartum period. Unilateral involvement is the rule but bilateral involvement can occur. (15).

Organism : streptococcus-most common(endocarditis),staph aureus(cutaneous), Bacillus(I.V), neisseria meningitides, Haemophilus Influenza. Onset is acute .

Clinical features- pain , ↓vision hypopyon , vitritis , bilateral involvement, endogenous fungal endophthalmitis develops slowly as focal or multifocal areas of chorioretinitis. (18).Granulomatous or nongranulomatous inflammation is observed with KP,

hypopyon, vitritis with cellular aggregate. Infection usually begins in choroids appearing as yellow white lesion with indistinct borders.

Candida most common organism. Occur in association with hyperalimentation, indwelling intra vascular line & IV drug use, major surgery or immunosuppression.

e) Post operative sterile Endophthalmitis(aseptic) :

i) Primary :

Onset –three to four days after surgery. Rarely weeks or months after intraocular surgery.

Causes : Acute

1. Due to surgery associated with vitreous manipulation
2. Retained lens material including cortex mixed with Vitreous
3. incarceration of iris, vitreous & lens material in corneoscleral wound
4. toxemia as a result of infectious disease like bacillary dysentery

If after weeks :

1. late rupture of anterior hyaloid membrane and vitreous adhesion to wound causing traction
2. degradation of implant materials
3. Dislocation of IOL
4. IOL pressure necrosis including UGH syndrome

Clinical features : Intense AC reaction ,hypopyon ,vitritis ,altered fundal reflex.

2) Secondary sterile post –operative endophthalmitis :

Causes : Endogenous factors

- ❖ Intraocular lens
- ❖ Foreign materials like cellulose ,conjunctival epithelium, glass ointment
- ❖ Chemicals –acetylcholine,alcohol, alpha chymotrypsin
- ❖ Drugs –preservative
- ❖ Dry pack sterilization of IOL's with ethyleneoxide

V) Lens associated uveitis :

i) Phacoanaphylactic endophthalmitis :

This is an immune response to lens protein released after injury to the lens capsule or after extracapsular surgery. Altered tolerance to lens protein leads to inflammation. which has an abrupt onset but may occasionally occur insidiously.

Clinical features : mutton fat KPs may appear on the cornea . Posterior synechia and dense flare and cells are present. HPE studies reveal Zonal granulomatous inflammation at the site of lens injury.

Phacoanaphylactic endophthalmitis has been described after trauma and IOL implantation following ECCE (3)

ii) Phacotoxic uveitis :

This is the term used to refer to the supposedly toxic effect of lens protein that enter the anterior chamber. It is really a less severe form of phacoantigenic endophthalmitis. An example is a mild to moderate inflammatory response following cataract surgery which is sometimes associated with hypopyon (38) (42).

iii) Phacolytic uveitis :

This condition involves an acute increase in IOP caused by clogging of the trabecular meshwork by lens protein and engorged macrophages. (9) (17)

Clinical features - Increased IOP, lack of KPs, refractile bodies in the aqueous and lack of synechiae.

VI) Tuberculosis associated uveitis :

TB is a chronic granulomatous infection by either bovine or human tubercle bacilli. Both direct infection and delayed hypersensitivity are implicated in the pathogenesis of tuberculous uveitis granulomatous and nongranulomatous anterior uveitis occurs. The diffuse miliary tuberculosis can involve the iris often producing an exudative iridocyclitis with hypopyon.

Clinical features : Chronic iridocyclitis –usually granulomatous but may occasionally be nongranulomatous with hypopyon in severe cases (11) Choroiditis –focal or multifocal, Retinal vasculitis –especially periphlebitis, Vascular occlusion, Dense vitritis, Papillitis

The patients experience a waxing and waning course with long term damage to Blood aqueous barrier accumulation of vitreous opacities and cystoid macular edema.

VII) Leptospirosis :

Zoonosis caused by spirocheate of the genus leptospira. Human beings are accidental hosts who acquire disease through contact with infected urine ,tissue or water. They invade human beings through abraded skin or mucous membrane. Once inside the host leptospires enter the bloodstream can penetrate tissues and are found in almost all organ with in 24 hours

Clinical manifestation :

Incubation period -2 to 26 days

Biphasic – first or leptrospiraemia phase is abrupt in onset & characterized by frontal & retro orbital headache, fever & chills. Conjunctival suffusion occurs on the third or fourth day.

Immune phase of disease begins 6 – 12 days of illness & corresponds to time when antibodies appear in serum .

Weils disease : Severe form of leptospirosis – jaundice, anaemia , azotemia , renal & hepatocellular dysfunction occurs on third to sixth day of infection .

Ocular findings : Conjunctival hyperaemia , scleral icterus, sch, retinal haemorrhages , optic neuritis can occur . Uveitis can occur in 10 – 40 % of patients which is a bilateral iridocyclitis with fine KP's , posterior synechiae , vitreous opacities, Hypopyon uveitis occurs in 13% of cases. (32,33,34)

VIII Masquerade Syndromes :

Comprises a group of disorders that occur with intraocular inflammation and are often misdiagnosed as a chronic idiopathic uveitis. It can be caused due to malignant disorder or non malignant disorders.

Malignant disorders :

Intraocular lymphomas, carcinoma metastatic to eye from lung, uveal melanoma, child hood carcinomas like retinoblastoma, leukemia, Juvenile xanthogranuloma and paraneoplastic syndromes.

Non – malignant disorders :

IOFB Retinal detachment, myopic degeneration, pigment dispersion syndrome, drug reaction to Rifabutin, Didanosine.

A high degree of suscipicion and a anterior chamber paracentesis is needed to clinch the diagnosis.

IX - IDIOPATHIC :

50% of patients with anterior uveitis have no aetiological factor established and these are termed as Idiopathic anterior uveitis. Not only they lack any systemic disease association characteristic of other anterior uveitis, they are also. HLA B27 negative. The pattern is one of a non-granulomatous uveitis with varying inflammatory response from mild iridocyclitis to hypopyon uveitis.

Review of Literature :

1. Alijandro Rodrigueres, et al – posterior segment ocular manifestation in patients with HLA B 27 associated uveitis.

The mean initial visual activity was 6/24 the anterior segment inflammatory reaction was non granulomatous in the great majority

of patients (90.4%), and was accompanied by an intense anterior chamber fibrinous reaction in (37.9%) patients. Hypopyon formation was present in 13.7% and occurred more commonly during a recurrent episode.

In 27.5%, secondary glaucoma developed. Posterior segment ocular manifestation were bilateral and asymmetric in 17.2% patients. Posterior clinical manifestation included severe vitritis (93%), papillitis in 83%, retinal vasculitis in 24% and 38% developed cystoid macular edema. The frequency of cystoid macular edema as a complication of uveitis may be attributed to the severity and the prolonged duration of the inflammatory activity.

2. Kearney et al, Clinical features and associated systemic diseases of HLA B 27 uveitis.

There was a male to female ratio of 1.5 :1. The median age at onset of uveitis was 32 years; 5% had then first attack after 55 years. Acute anterior uveitis was noted in 87% and non acute inflammation was noted in 13% ocular involvement was categorized as unilateral

or unilateral alternating in 93% & 7% had bilateral concurrent disease.

The median duration of attack was six weeks, and the median number of recurrences was 3.32 cataracts were associated with posterior synchiae (p=0.03) increased intraocular pressure was seen in 23% cataracts were seen in 30%. An HLA B 27 associated systemic disease was present in 58% of patients. 30% had a family history of spondylarthropathy.

3. D' Alessandro et al – Anterior uveitis and hypopyon

7.1% of anterior uveitis patients developed hypopyon. The hypopyon occupied from 5% - 15% of anterior chamber. The patients ranged in age from 10-63 years (mean – 37 years) and there was no predilection for either sex.

In all cases hypopyon was unilateral and in no cases were thee large granulomatous keratic precipitates. Visual activity at the time of the hypopyon was 20/200 or less. 82% of patients with acute anterior uveitis who developed hypopyon were HLA B 27 positive.

33.33% developed posterior subcapsular cataracts. The incidence of hypopyon in HLA B 27 positive group ($p < 0.003$).

4. Rathinam et al, uveitis associated with an epidemic outbreak of leptospirosis.

Uveitis associated with leptospirosis may manifest as unilateral or bilateral uveitis, anterior uveitis, or panuveitis. Awareness of this disease in endemic area is important in order to differentiate it from other uveitis entities, especially in young male patients in whom other immunologic uveities are also common.

Complications :

1. Cataract - chronic or recurrent inflammation

- steroid induced

2. Glaucoma – secondary

-open

-closed

-neovascular

- Open – trabeculitis

  - Clogging of Trabecular Meshwork with inflammatory cells , plasmoid acqeous

- Closed – posterior synchiae

  -pupillary block

  -Periphral anterior synchiae

- Neovascular – chronic irido cyclitis

Hypotony - temporarily decreases aqueous production by inflammed ciliary body.

- permanent ciliary body damage by cyclitic

membrane formation & contraction with detachment.

- choroidal effusions

Cystoid macular edema is the commonest cause of visual loss.

Vitreousopacification & vitritis

Retinal detachment – tractional or rhegmatogenous

Band Keratopathy .

Investigations:

1. TC,DC, ESR, RF,ANA, CRP –seronegative spondylo arthropathy
2. Mantoux, CXR –TB
3. X Ray sacroiliac joint-ankylosing spondylitis
4. Hand X Ray
5. Aqueous tap
6. Vitreous biopsy
7. FFA-cystoid macular edema, srnvm, Disc leakage, late staining of retinal vessels, Retinal vascular capillary dropout, RPE perturbation
8. USG B Scan –for posterior segment evaluation
   - -complicated cataract
   - -small pupils
   - -severe vitritis
   - -RD
9. Newer Diagnostic Tests
   - -PCR-TB
   - -Direct flourescein antibody test

THERAPY FOR HYPOPYON UVEITIS

Observation :

For development of complications .

For change in the appearance / severity /progression

MEDICAL MANAGEMENT OF UVEITIS

Medical therapy includes topical or systemic cortico steroids & topical cycloplegics . Immunosupressive therapy may be required in patients with severe uveitis of unresponsive to corticosteroid therapy or in patients with severe corticosteroid induced complications. Treatment should be tailored as specifically as possible to the individual patients & adjusted according to response.

1. Cycloplegics :

Topical cycloplegic agents are beneficial for breaking or preventing the formation of posterior synechiae & providing relief of photophobia secondary to ciliary spasm. The stronger the inflammatory reaction, the stronger or more frequent is the dosage of cycloplegic. Short acting cycloplegics are used in acute anterior uvietis, these allow the pupil to be mobile .

2. Corticosteroids :

They are the mainstay of uveitis therapy. Because of their potential side –effects they should be reserved for specific disease Indications :

-Treatment of active inflammation in the eye

-prevention or treatment of complications such as cystoid macular edema .

-reduction of inflammatory infiltration of the retina, choroid or optic nerve .

These agents should be used only in cases where the benefits of therapy outweigh the risks of the medications.

If steroid therapy is needed for longer than 2-3 weeks then dosage should be tapered before stopping. The dosage should be increased during surgical intervention to prevent exacerbation of the uveitis postoperatively.

a) Topical :

Indication : Anterior uveitis, vitritis or macular edema in patients with aphakia or pseudophakia.

They do not reduce chronic flare in AC, hyalinized KP or pigment in the aqueous caused by dilatation. Rimexolone & fluoromethalone have less of ocular hypertension effect.

b) Periocular :

Indications :

-When a more posterior effect is needed

-noncompliant patient

-poorly responsive to topical or systemic therapy

-performed with either a transeptal or sub-tenon's approach

-with a sub-tenon's injection , a 25-gauge needle 5/8 " needle is used .

-contra indicated in infections uveitis, scleritis

Complications :

scleral thinning, scleral perforation

c) Systemic administration :

Oral or IV therapy may supplement or replace other routes of administration.

Indications :

- Chronic bilateral uveitis that threatens vision
- Topical steroids are insufficient.
- Systemic disease also requires therapy.

Complications :

Increased appetite, weight gain, peptic ulcers, sodium & fluid retention, osteoporosis, bone fractures, aseptic necrosis of hip, hypertension, diabetes, menstrual irregularities, mental status changes, exacerbation of systemic infections, impaired wound healing.

3. Immunomodulating & Immunosupressive agents :

- these agents modulate the immune system
- these agents are nonsteroid anti inflammatory agents & immuno suppressive agents.

a. NSAIDS :

Topical NSAIDS may be useful in the treatment of postoperative inflammation & cystoid macular edema.

b. Cytotoxic agents & antimetabolites :

they act by killing the rapidly dividing clones of lymphocytes that are responsible for inflammation. Since the benefits outweigh the risks in many cases they are considered the first line of therapy in certain cases.

Indications :

- Vision threatening intraocular inflammation.
- reversibility of the disease process
- lack of response to corticosteroid treatment
- reversibility of disease process
- contra indication of corticosteroid treatment because of systemic problems or intolerable side effects.
- Behcet's , rhematoid necrotizing sclerouveitis.

Relative indications :

- if corticosteroids fail to control the inflammation.
- intermediate uveitis, retinal vasculitis, chromic iritocyclitis .

Complications :

Future malignancies like leukemia , lymphoma, soft-tissue tumors , opportunistic infections .

- Absence of inflection
- Absence of heamatologic contra indications
- Objective evaluation of the disease process
- Informed consent

Alkylating agents :

Cyclophosphamide – 1 - 2 mg / kg /day

Chlorambucil – 2 mg /day then increased to 8–12 mg / day

Antimetabolites :

Azathioprine – 50 -150 mg / kg

Methotrexate – 7.5 – 25.0 mg oral or 1m

Cyclosporine A – naturally occurring compound produced by fungi .

- specific effect on immune function
- inhibits T-cell activation & recruitment, dosage is 2.5 – 5.0 mg / kg, side effects are nephrotoxicity & hypertension, se-creatinine values should be monitored, sustained release implants are available

Recent advances in management :

1. Colchicine : decreases PMN cell migration & phagocytosis.
   - This is not used in active inflammation but used

prophylactically in patients with multiple attacks, dose is 0.6 mg BD orally, side effects are bone marrow depression & aplastic anemia.

2. Bromocriptine : prolactin increases immune response by stimulating lymphocyte activation, it is a dopamine against which decreases prolactin & immune response, dose is 4 – 5 mg / kg / Day

2. FK506 – isolated from streptomyces suppresses formation of mRNA. Dose is 0.10 – 0.15 mg / kg / day, it is nephrotoxic, ophthalmoplegia can occur.
3. Mycophenolate mofetil : Inhibits DNA synthesis.
4. Rapamycin : Macrolide immunosuppressant.

- It prevents expression of IL -2,4,6

It blocks proliferative signals of transaction in T-cells, Dose is 0.01 mg /kg / day. It causes myocardial toxicity, GI vasculitis

5. Monoclonal antibody therapy :

- anti T-cell monoclonal Ab, anti IL- 2 receptor Ab.

Oral tolerance : 30 mg of retinal s-antigen is given orally 3 times / week, the patients develop tolerance rather than immune response.

Plasmapheresis : Behcet ‘s disease

Levamisole : Immune stimulator.

II - Surgical therapy :

Diagnostic – Anterior chamber aspiration

- Vitreous biopsy

Reparative –undertaken after control of inflammation

1. Cataract surgery – complicated cataract
   - injury to lens capsule with leaking lens matter
   - posterior segment repair needed

Surgery is problematic due to miotic pupil , posterior synechiae which should be managed by multiple sphincterotomies & synechiolysis . Cortical cleanup should be meticulous , surface modified IOLS should be implanted .

2. Glaucoma surgery– after control of inflammation & IOP medically.
3. The following procedures are recommended.
   i. Secondary angle closure – papillary block can be relieved by laser peripheral iridectomy if permanent peripheral anterior synechiae has not formed .
   ii. Secondary open angle – standard trabeculectomy has a high failure rate . Use of adjuvant metabolites like 5 - flurouracil & mitomycin-c increases success rate. Other

recommended procedures are Trabeculodialysis, a modified goniotomy, laser sclerostomy are recommended procedures. In cases where other surgeries have failed aqueous drainage devices can be used.

iii. Pars plasna vitrectomy –endophthalmits ,cyclitic membrane

iv. Retinal detachment surgery / Pupillary reconstruction

v. Scleral buckling

AIM OF THE STUDY

A prospective descriptive study was undertaken to study all the cases of uveitis (39 cases) with hypopyon that presented to the out patient department of Govt. Rajaji Hospital between June 2004 and August 2005 using a standard protocol and compared with the pattern of hypopyon uveitis in other studies.

1. To analyse the spectrum of cases of uveitis with hypopyon presenting at a tertiary care hospital with regard to

    a. patient profile,

    b. clinical presentation and

    c. aetiology

and to compare results with previous published studies.

MATERIALS AND METHODS

39 cases of uveitis with hypopyon were included in this prospective study conducted in our department between June 2004 and August 2005.

INCLUSION CRITERIA:

Any case of uveitis with significant anterior chamber inflammatory reaction and hypopyon without any primary ocular infection was included in the study. All the cases of endophthalmitis with hypopyon were included in the study except that caused by penetrating or perforating injuries of globe.

EXCLUSION CRITERIA :

Cases with infective corneal pathology or corneal trauma with hypopyon were excluded from the study thereby excluding exogenous infective aetiologies.

The questionnaire included the demographic details, socio economic status and a detailed ocular examination including slit lamp biomicroscopy and Fundus examination with 90D examination and indirect ophthalmoscopy. The patients were subjected to relevant laboratory investigations, a clinical diagnosis made and appropriate treatment started. All patients were followed up and during every follow up visit a detailed ocular examination was made.

The questionnaire included

1. Age
2. Sex
3. Address
4. Laterality
5. Presenting complaints in detail
6. Leading questions regarding various etiological factors
    a) HLA- B27 related iridocyctitis
    b) Viral keratouveitis
    c) Leptospirosis
    d) Pet (Toxoplasmosis / Toxocariasis)
    e) Infective foci elsewhere in the body

f) Contact with tuberculosis

g) Symptoms related to genitourinary system, gastro intestinal system, CNS, respiratory system and skin diseases.

7. Past history of trauma, eye inflammation and prior visual loss

8. Treatment history

9. Ocular examination included

a) Visual activity (best corrected)

b) Intra ocular pressure at presentation

c) Detailed slit lamp examination : types of keratic precipitates, anterior chamber reaction, colour and the amount of hypopyon, iris nodules, synechiae lens changes and changes in anterior vitreous face.

d) Fundus examination with direct and indirect ophthalmoscopy, 3 mirror examination.

10. Systemic examination to R/o any associated systemic disease and medical, Rheumatological and orthopaedic opinion where ever needed.

Subsequently a tailored laboratory investigation was carried out. The investigations included

1. Total leucocyte count
2. Differential count
3. Erythrocyte sedimentation rate
4. Mantoux test
5. VDRL
6. ELISA for HIV I & II
7. RA factor
8. IgM for leptospirosis
9. Ultrasound B scan
10. FFA
11. X ray chest, Sacroiliac spine

The final aetiological diagnosis was made based on the clinical features, relevant investigations and systemic evaluation by medical specialists.

HLA B 27 related uveitis was diagnosed mostly on the basis of clinical presentation with features of low back ache history of

significant joint pain ie., joint pain involving larger joints lasting for a period of time, fibrinous hypopyon uveitis with multiple posterior synechiae and other eye showing evidence of old uveitis and those who were diagnosed as HLA B27 patients by the Rheumatology and medicine department. Endogenous endophthalmitis was diagnosed mostly on the acute presentation with hypopyon, USG evidence of vitreous exudates and culture positivity.

RESULTS AND COMPARATIVE ANALYSIS

A prospective study involving 39 cases of hypopyon uveitis presenting at the eye department of Govt. Rajaji Hospital was done and the following results obtained.

1. Aetiology of hypopyon uveitis

Aetiology	Number	%
HLAB 27 uveitis	9	28.20
Leptospirosis	4	10.25
Idiopathic	7	17.94
Herpes keratouveitis	5	12.82
Exogenous endophthalmitis	2	5.12
Endogenous endophthalmitis	1	2.56
Sterile endophthalmitis	4	10.25
Lens induced uveitis	4	10.25
TB associated uveitis	2	5.12
Leukemia(Masquerade syndrome)	1	2.56

The most common cause of hypopyon uveitis in this study was uveitis associated with HLAB 27 diseases accounting for 9 cases(28.2%). The other cases were leptospirosis 4 cases(10.25%), Herpes keratouveitis 5 cases(12.8%), exogenous endophthalmitis 2

cases(5.1%), endogenous endophthalmitis 1 case(2.5%), sterile endophthalmitis 4 cases(10.2%), lens induced uveitis 4 cases(10.25)%). TB associated uveitis 2 cases(5.1%), leukemia 1 case (2.5%) and the rest of the 7 cases(17.9%) accounting for Idiopathic cause.

2. Demographic characteristics :

a) Age Distribution

Age	Number	%	HLAB27	%
0 -9	1	2.56	0	-
10-19	1	2.56	0	-
20-29	5	12.82	1	11.11
30-39	18	46.15	8	88.88
40-49	5	12.82	-	-
50-60	7	17.94	-	-
> 60	2	5.12	-	-

Most cases of hypopyon uveitis were between II and V decade of life with maximum of 18 cases (46.15%) in the 30-39 age group. The commonest cause of hypopyon uveitis in this study was HLAB27 & Leptospirosis in middle age and lens induced and TB associated uveitis in old age.

2b. Sex Distribution

Sex	Total	%	HLAB27	%
Male	22	56.41	6	66.66
Female	17	43.58	3	33.33

22 (56.41%) cases of hypopyon uveitis occurred in males while17 cases (43.58%) were females. In HLA B-27 uveitis, 6 cases (66.66%) occurred in males and 3 cases (33.33%) occurred in females Kearney et al observed in his study a male to female ratio of 60 : 40 in HLA B27 associated uveitis.

2C - Laterality

Laterality	Total	%	HLAB27	%
Unilateral	38	97.43	9	100
Alternating Unilateral	2	5.12	2	22.22
B/L uveitis with U/L hypopyon	1	2.56		

2d) Unilateral hypopyon uveitis

Side	Total	%	HLAB27	%
Right	18	46.15	5	55.55
Left	21	53.84	4	44.44

38 cases (97.43%) of hypopyon uveitis were U/L with the left eye (53.84%) more commonly affected than the R eye (46.15%). 1 case (2.56%) presented with Bilateral uveitis with unilateral hypopyon. 9 cases (100%) of HLAB27 uveitis presented with hypopyon uveitis and 55.55% affected the Right eye and 44.44% the Left eye.

2 e) Socio economic Status :

E.Status	Total	%	HLAB27	%
High	4	10.25	23	34.33
Low	35	89.75	44	65.67

35 cases (89.74%) of hypopyon uveitis belonged to the low socio economic status. An income of less than Rs. 500 per month was considered low.

3. Symptoms at presentation :

Symptoms	No.	%
Pain	37	94.87
Redness	37	94.87
Photophobia	21	53.84
Floaters	7	17.94
Defective vision	37	94.87

The most common symptoms with which the patient presented in this study are pain 94.87 %, redness 94.87 % and defective vision 94.87%

4. Hypopyon Uveitis associated with other systemic diseases.

Disease	No.	%
HT	3	7.69
DM	2	5.12
Tuberculosis	2	5.12
GI	1	2.56

Systemic diseases associated with hypopyon uveitis were diabetes mellitus (5.12%), Hypertension (7.69%), tuberculosis (5.12%) and Gastrointestinal disease (2.56%)

5. Treatment used at presentation

Treatment modality	No.	%
Topical steroids	5	12.82
Subconjunctional steroids	2	5.12
Systemic steroids	3	7.69
Cycloplegics	4	10.25
Native medications	1	2.56
No treatment	32	82.05

Majority (82.5%) of the patients had not used any form of treatment at presentation. The commonest treatment modality used was topical steroids 12.82 % and cycloplegics 10.25%.

6. Mode of Onset

Onset	Total	%	HLAB27	%
Acute	35	89.74	8	88.88
Insidious	3	7.69	1	11.11
Chronic	1	2.56	-	-

89.74 % of hypopyon uveitis had an acute onset while 7.69 % had an insidious onset and 2.56 % had a chronic onset. 88.88% of HLA

B-27 uveitis had an acute onset and 11.11 % of them had an insidious onset.

7. Course of the disease

Course	Total	%	HLAB27	%
Acute	27	69.23	8	88.88
Subacute	7	20.51	-	-
Chronic	1	2.56	-	-
Recurrent	4	10.25	2	22.22

69% of HU had an acute course, 20.51% had an subacute course, 10.25% had a recurrent attack and 2.56% had a chronic course. 88.88% of HLA B27 hypopyon uveitis had an acute course and 22.22 % had a recurrent course.

8. Location :

Location	Total	%	HLA B27	%
Anterior uveitis	22	56.41	7	77.77
Panuveitis	17	43.58	2	22.22

22 cases (56.41%) of hypopyon uveitis presented with anterior uveitis and 17 cases (43.58%) had panuveitis. In HLAB27 uveitis HU presened as anterior uveitis in 7 cases (77.77%) and panuveitis in 2 cases (22.22%).

9. Visual activity at presentation

Visual Acuity	Total	%	HLAB27	%
6/6 – 6/12	5	12.82	-	-
6/18 – 6/24	18	46.75	6	66.66
6/36 – 6/60	7	17.94	3	33.33
5/60 – 1/60	2	5.12	-	-
< 1/60	7	17.94	-	-

The visual acuity at presentation ranged from 6/12 to PL 18 cases (46.75%) had V/A between 6/18-6/24; 7 cases (6/36-6/60), 5 cases (12.82%), 2 cases (5/60-1/60) and 7 cases (17.94%) < 1/60. In HLA B27 hypopyon uveitis 6 cases (66.66%) had V/A between 6/18-6/24 and 3 cases (33.33%) had 6/36 – 6/60 at presentation.

10. IOP at presentation

Tension	Total	%	HLAB27	%
< 10 mm	1	5.12		
10-21	28	71.79	7	77.77
> 21	10	25.64	2	22.22

Majority of eyes with hypopyon uveitis had IOP between (10-21) ie. 28 cases (71.79%) and 10 cases (25.64%) > 21mm Hg and 1 case (5.12%) < 10mm Hg. In HLAB27 hypopyon uveitis 7 cases (77.77%) had IOP between 10-21 mm Hg and 2 cases (22.22%) > 21mm Hg. Kearney et al has observed that 23% of cases in HLAB27 hypopyon uveitis had IOP > 21mm Hg.

11. Level of Hypopyon

Level(mm)	Total	%	HLAB27	%
≤ 0.5	5	12.82	8	88.88
1mm	29	74.35	1	11.11
2.0	2	5.12		
≥ 2.5	3	7.69		

34 cases (87.17%) with hypopyon uveitis had hypopyon < 1.0 mm and 5 cases (12.82%) had > 2mm of hypopyon.

12. Colour of hypopyon

Colour	Total	%	HLAB27	%
White	28	71.79	6	66.66
Yellow	10	25.64	3	33.33
Blood tinged	1	2.56		

28 cases (71.79%) of HU had white hypopyon and 10 cases (25.64%) had yellow colour, and 1 case (2.56%) had blood tinged hypopyon which was due to viral keratouveitis. 6 cases (66.66%) of HLA B27 uveitis had white hypopyon and 3 cases (33.33%) had yellow hypopyon.

13. Lens changes in hypopyon uveitis

Lens	Total	%	HLAB27	%
Clear lens	21	53.84	6	66.66
Senile cataract	3	7.69	-	-
Complicated cataract	10	25.64	3	33.33
PCIOL	5	12.82	-	-

21 cases (53.84%) of hypopyon uveitis had clear lens at presentation and 10 cases (25.64%) had complicated cataract. In HLA B-27 uveitis 6 cases (66.66%) had clear lens and 3 cases (33.33%) had complicated cataract. Kearney et al has observed that complicated cataract occurred in 30% of HLA B27 uveitis.

14. Posterior segment finding in hypopyon uveitis

Posterior segment	Total	%	HLAB27	%
Vitritis	15	23.3	3	33.33
Disc edema	1	2.56	-	-
Vasculitis	2	5.12	-	-
Cystoid macular edema	9	28.20	3	33.33

Posterior segment involvement in cases of Hypopyon uveitis was 15 cases (23.30%) of vitritis, 9 cases (28.20%) of CME, 1 case (2.56%) of Disc edema, 2 cases (5.12%) of vasculitis. In HLA B27 Hypopyon uveitis 3 cases (33.33%) had CME.

Rodriguerez et al observed that 93% of cases had vitritis and 38 % had cystoid macular edema.

15. Complications

Complications	No.	%
Complicated cataract	10	25.64
Recurrent uveitis	2	5.12
Secondary glaucoma	10	25.64
Steroid induced glaucoma	-	-
Occlussio pupillae	3	7.69
Seclusio pupillae	2	5.12
Choroidal detachment	-	-

The commonest complication of Hypopyon uveitis was development of complicated cataract in 10 cases (25.64%) and secondary glaucoma in 10 cases (25.64%).

SUMMARY OF RESULTS

The results of the study are :

1. HLA B 27 uveitis (28.2%) was the commonest cause of hypopyon uveitis in our study.
2. Most cases of hypopyon uveitis were between 30-39 age group (18 cases) 46.15%
3. 56.14% cases of hypopyon uveitis occurred in males and 43.58 % occurred in females
4. 97.43% of hypopyon uveitis were unilateral with left eye more commonly affected than Right eye. 100% of HLA B 27 uveitis presented with unilateral hypopyon
5. 94.87% of hypopyon uveitis presented with pain, redness and photophobia
6. 82.05% of patients had not used any form of treatment at presentation.
7. 89.74% of hypopyon uveitis had an acute onset, 88.88%of HLA B 27 uveitis had an acute onset.
8. 46.75% had V/A between 6/18 – 6/24. 66.66 % of HLA B 27 had V/A between 6/18 – 6/24.

9. 56.41% of hypopyon uveitis presented with anterior uveitis and 43.58 % had panuveitis. In HLA B 27 hypopyon uveitis presented as anterior uveitis in 77.77% and panuveitis in 22.22%

10. 25.64 % of hypopyon uveitis had IOP > 21 mm Hg.

11. 87.17 % of hypopyon uveitis had hypopyon < 1.00 mm at presentation.

12. 25.64 % of hypopyon uveitis had complicated cataract. In HLA B 27 uveitis 33.33% had complicated cataract.

13. 56.14% of hypopyon uveitis had posterior segment findings of vitritis, CME, vasculitis.

14. Most common complication of hypopyon uveitis was complicated cataract (25.64%) and secondary glaucoma in (25.64%)

DISCUSSION

Hypopyon Uveitis is a feature of any hyperacute severe form of uveitis, but is characteristic of some specific aetiologies like Behcet's disease.

Our study was compared to Kearney et al, D Alessandro et al and Rodriguerez et al

- HLA B 27 (28.20%) uveitis was the commonest cause of Hypopyon Uveitis in our study. This is comparable to study of D 'Allessandro et al who has observed a similar distribution.
- 46.15% of Hypopyon Uveitis presented in the 30-39 age group with 56.41% o them being males. 88.88% of HLA B 27 related hypopyon uveitis presented in the 20-50 age group and there was a male preponderance of sex distribution (66.66 : 33.34). Kearney et al observed in his study 85% of cases HLA B 27 were in the 20-50 age group and a male to female ratio of 60 : 40, which is comparable to our study.

- Hypopyon Uveitis presented unilaterally in 97.43 % of cases. In HLA B27 presentation was unilateral in 100 % of cases.
- Most of the cases (89.74%) were from low socio-economic status.
- Course of the disease in hypopyon uveitis was acute in 89.74% and in HLA B27 uveitis it was acute in 88.88%.
- The visual acuity at presentation in hypopyon uveitis was between (6/12 – 6/24) in 46.75% and In HLA b27 uveitis the visual acuity was between 6/18 – 6/24 in 66.66 % of cases.
- Increased Intraocular pressure was present in 25.64% of hypopyon uveitis and in HLA B 27 uveitis 22.22% of cases had increased IOP. Kearney et al observed a increased IOP in 23% in his study.
- Complicated cataract occurred in 25.64% of cases in hypopyon uveitis. In HLA B 27 uveitis 33.33% were complicated by cataract. Kearney et al observed an evidence of 30% of complicated cataract in HLA B 27 uveitis.

- Posterior segment findings in Hypopyon Uveitis were predominantly vitritis (23.30%) and cystoid macular edema (28.2). The 33.33% of cases of HLA B27 uveitis had CME. Rodriguerez observed an incidence of 38% cystoid macular edema in HLA B27 uveitis with hypopyon.
- The commonest complication of hypopyon uveitis in this study was complicated cataract (25.64%) and secondary glaucoma (25.64%).
- The other causes of Hypopyon uveitis in our study were

  4 cases (10.25%) due to leptospirosis, 5 cases (12.82%) due to Herpes karatouveitis, 2 cases (5.12%) due to exogenous endophthalmitis, 1 case (2.56%) of endogenous endopthalmitis, 4 cases (10.25%) of sterile endopthalmitis, 4 cases (10.25%) of lens induced uveitis, 2 cases (5.12%) of tuberculosis uveitis and 1 case (2.56%) of leukemia (Masquerade syndrome) for 7 cases (17.94%) cause could not be identified.

CONCLUSION

Hypopyon uveitis refers to certain specific uveitic entities that are characterized by a sterile collection of leukocytes and variable amount of fibrin indicating of severe inflammatory response. In the west the common cause of hypopyon uveitis are Behcet's disease but in our subpopulation due to varied cultural, hygienic characteristics the aetiologies of hypopyon uveitis is expected to be different.

This prospective study was basically a descriptive study analyzing 39 cases of hypopyon uveitis that presented to the eye department of Govt. Rajaji Hospital. In our study the commonest causes of hypopyon uveitis was found to be HLA B27 related uveitis. There was a male preponderance in cases which presented with hypopyon uveitis most commonly the Left eye was affected. The most common mode of presentation was unilateral and of acute onset. The age of presentation was between II and V decade of life. Most common posterior segment finding was cystoid macular

edema. The most common complication was complicated cataract and secondary glacucoma.

HLA B 27 related hypopyon uveitis presented most commonly between the 3rd and 4th decade of life. Males were more commonly affected and all the cases were unilateral with acute onset in most cases. The most common complication was complicated cataract and most common posterior segment finding was cystoid macular edema.

This study is a descriptive study and has its own limitations. For example HLA B 27 was diagnosed based on clinical features and that were diagnosed as HLA B 27 disease by the Rheumatology and medical department.

The results of out study are comparable with other studies.

BIBLIOGRAPHY

1. Aaberg TM Jr. Flynn Hw Jr et al, Nosocornial acute onset postoperative endophthalmitis survey. Ophthalmology 1991; 98 : 227-238.
2. Abraham et al. Leukemia hypopyon. J. Paed ophthalmol & stabis 1982; 18 : 42-44.
3. Apple DJ et al. Phacoanaphylatic endophthalmitis associated with extra capsular extraction with posterior chamber intra ocular lens. Arch ophthalmol 1984 ; 102 : 1528 – 1532.
4. Craig HJ et al. Ocular tuberculosis. Surg. Ophthalmol 1993; 38 : 229 – 256.
5. Biswas et al. Immunopathology of uveitis. Indian J ophthalmol 1998 ; 70 : 11-25
6. Duke elders, Perkins ES. System of ophthalmology, vol. VIII, Henry kimpton, 1966 : 757 – 758.
7. D' allexandro et al. Anterior uveitis and hypopyon. Am J ophthalmol 1991 ; 112 : 317-321.

8. Duke elders, Perkins ES. System of ophthalmology, vol. IX Henry kimpton, 1966 : 346.
9. Duke elders, Perkins ES. System of Ophthalmology, vol. IX, Henry kimpton, 1966 : 508.
10. Duke elders, Perkins ES. System of ophthalmology, vol. IX, Henry kimpton, 1966 : 891.
11. Galezowski N et al. Hypopyon uveitis and tuberculosis. Rev – Med – Interne 1966 ; 17 : 858 – 859.
12. Hemady et al. Immunosuppressive drugs in ocular inflammatory diseases. Surv ophthalmol 1991 ; 35 : 369 – 385.
13. Iijima S et al. Psoriatic arthritis and hypopyon iridocyclitis. Dermatology 1996; 193 : 295 – 299.
14. International ophth clinics. Vol. 36, No.1, winter 1996 : 127 – 134.
15. Jakobiec FA, Albert DM. Principles and practice of ophthalmology, WB Saunders, 1994 : 415.
16. Jakobiec FA, Albert DM. Principles and practice of ophthalmology, WB Saunders, 1994 : 2265.

17. Jakobiec FA, Albert DM. Principles and practice of ophthalmology, WB Saunders, 1994 : 2218.
18. Jakobiec FA, Albert DM. Principles and practice of ophthalmology, WB Saunders, 1994 : 3120-3125.
19. Jacobs DS et al. Acute uveitis associated with Rifabutin use in patients with human immuno deficiency virus infection. Am J ophthalmol 1994 ; 118 : 716 – 722.
20. Jakobiec FA, Albert DM. Principles and practice of ophthalmology, WB Saunders, 1994 : 1018-1026.
21. Kearney et al. Clinical features and associated features of HLA B 27 Uveitis. Am J ophthalmol 1997; 121 : 47-56.
22. Nussenblatt RB, Whitcup SM, Palestine AG. Uveitis – Fundementals and clinical practice, Mosby, 1996 : 69-78.
23. Narsing AR, Blackman HJ, Basic and clinical science course – Intraocular inflammation and uveitis (section 9), American academy of ophthalmology, 1997 – 98 : 41-58.
24. Nussenblatt RB, Whitcup SM, Palestine AG. Uveitis – Fundamentals and clinical practice, Mosby, 1996 : 334 – 350.

25. Nussenblatt RB, Whitcup SM, Palestine AG. Uveitis – Fundementals and clinical practice, Mosby, 1996: 390.
26. Narsing AR, Blackman HJ, Basic and clinical science course – Intraocular inflammation and uveitis (section 9) American academy of ophthalmology, 1997 ; 112.
27. Nozik RA, Smith RE. Uveitis- A clinical approach to diagnosis and management, William and Wilkins, 1989 : 198.
28. Nozik RA, Smith RE. Uveitis- A clinical approach to diagnosis and management, William and Wilkins, 1989 : 172-179.
29. Oster et al. Reiter's disease and uveitis. Am J Ophthalmol 1971 : 986 – 991.
30. Ophthalmology clinics of North America, March 1999 ; 12:54.
31. Rodriguerez A et al. Posterior segment manifestations of HLA B 27 Uveitis. Ophthalmol 1994 ; 101 : 1267 – 1274.

32. Rathinam et al. Leptospiosis. Journ of TNOA. 1997 Dec : 1-4

33. Rathinam et al. Uveitis associated with outbreak of leptospirosis. Am J. Ophthalmol 1997 ; 124 ; 71-79

34. Robert MS et al. Leptospial uveitis. Arch ophthalmol 1959; 61 : 633 – 639.

35. Richard R et al. Clinical laboratory Medicine. 1984: 156 – 157.

36. Rothova A et al. Clinical features of acute anterior uveitis. Am J ophthalmol 1987 ; 103 : 137-145.

37. Tasman W, Jaegar EA. Duanne's clinical ophthalmology, JB Lippincott, 1994; Vol. 4, chapter 39 : 2.

38. Tasman W, Jaeger EA. Duanne's clinical ophthalmology, JB Lippincott, 1994; Vol. 4, Chapter 41 : 6.

39. Wakefield et al. Acute anterior uveitis and HLA B 27. Surv. Ophthalmol 1991 ; 36: 223 – 232.

40. Winward KE, Flynn. HW Jr et al, Postoperative propionibacterium endopthalmitis. Ophthalmology : 1993; 100 : 447 – 451.

41. Winter FC et al. Oculopathology of Behcet's disease. Am J. ophthalmol 1966 ; 62: 257-262.

42. Wirostrko et al. Lens induced uveitis. Arch ophthalmol 1967 ; 78 : 1-7.

PROFORMA

AETIOLOGY OF HYPOPYON UVEITIS

Name : Date : Laterality - Right eye

Age : MR No. Left eye

Sex : Both eyes

Occupation : Case No.

Social Status :

History	**Right Eye** Duration	**Left Eye** Duration
Pain		
Redness		
Photobhobia		
Floaters		
Defective vision		
HLA B 27		
LBA		
Joint pain		
Swelling, redness of joint		
Fever,		
Skin lesions		
BEHCET's		
Recurrent oral ulcers		
Skin lesions – acneiform		
Recurrent genital ulcers		
Recurrent genital ulcers		

Recurrent ocular inflammation

Recurrent arthritis.

Intra ocular tumor :

White reflex

Proptosis

Pet animal

Pica

Infective foci (endo endoptitis)

Caries tooth

Non healing ulcer

Pulmonary infection : Expectoration

Genito urinary foci

Pyuria

Leptospirosis - Headache

Myalgia

Fever

Conjunctival suffusion

Systemic disease : HT

Diabetic

TB

Leprosy

Others

Treatment History

Topical

Subconjunctival		
Subtenon		
Systemic		
Previous attack	Yes	No
Vision corrected	R	L
Tension	R	L
S/L		
Conjunctiva		
Congestion		
Nodules		
Cornea		
Sensation		
Epithelium		
Stroma		
Endothelium		
AC		
Flare		
Cells		
Haemorrhage		
Hypopyon (mm)		
Iris		
Colour pattern		
Posterior synechiae		
PAS		
Nodules / granulomas		
Vessels		

Pupil :

Lens → Normal
Lens → senile cataract
Lens → Complicated cataract

Fundus — Media
Vitreous
Optic disc
Vessels
Retina
Choroid
Periphery

Lab tests :

Hg
ESR
TLC
DLC
VDRL — Chest, Sacroiliac, hand X ray
Mx — Others
HIV
USG
FFA — Diagnosis
Ig MAT (lepto)

MASTER CHART

S. No.	Name	Age	Sex	Course	Location	Laterality	V\A RE	V\A RE	Height	IOP RE	IOP LE	KPs	Cataract	Post segment	Lab findings	Diagnosis
1	Ranjitham	63	F	AC	Pan	U	PL+	6/18	1.0	50.6	17.3	Na		Vit		PU
2	Kumaran	37	M	AC	Ant	U	6/24	6/9	1.0	23.8	17.3		+	CME		HLA
3	Karuppiah	50	M	AC	Ant	U	6/9	6/18	1.0	17.3	14.6			CME		LEP
4	Rajapandi	37	M	AC	Ant	U	6/12	6/6	0.5	15.9	14.6			CME		IP
5	Meenakshi	61	F	AC	Ant	U	6/6	HM	2.0	14.6	57.6					PU
6	Parvathi	35	F	AC	Ant	U	6/6	6/36	1.0	14.6	21.9				JP	HLA
7	Sarasu	48	F	AC	Pan	U	6/24	6/6	1.0	14.6	14.6		+	Vit	Ig M	LEP
8	Pandian	39	M	AC	Ant	U	6/60	6/12	1.0	17.3	23.8			Vas		IP
9	Soundaram	61	F	AC	Ant	U	6/18	6/18	1.0	17.3	18.4					SE
10	Manian	57	M	AC	Ant	U	CFCF	6/18	2.5	46.9	14.6		+			PU
11	Selvi	34	F	AC	Ant	Alt U	6/9	6/18	1.0	14.6	12.2			CME	LBP	HLA
12	Selvam	51	M	SA	Pan	U	6/18	6/6	1.0	15.9	14.6			Vit	IgM	LEP
13	Dhanam	27	F	AC	Pan	U	6/12	6/18	1.0	14.6	15.9			CME		IP

14	Rajeshwari	60	F	AC	Pan	U	6/9	HM	1.0	57.6	17.3			Vit,C		PU
15	Sahul hameed	31	M	REC	Pan	U	6/36	6/9	1.0	12.2	14.6			Vit	JP	HLA
16	Raghavan	35	M	AC	Ant	U	6/9	6/24	1.0	17.3	12.2		+	CME	IgM	LEP
17	Lakshmi	25	F	AC	Ant	U	6/6	6/24	1.0	17.3	12.2			Disc		IP
18	Chinnavel	54	F	AC	Pan	U	PL+	6/24	3.0	12.2	14.6			Vit		PIE
19	Ahmed	31	M	AC	Ant	U	6/24	6/6	1.5	14.6	14.6			CME	JP	HLA
20	Rajan	32	M	REC	Pan	U	6/6	6/24	1.0	24.4	17.3	G		Vas	VC	HZKU
21	Murugesan	22	M	SA	Ant	U	6/9	3/60	1.0	12.2	13.6		+	CME		IP
22	Rasuthevar	57	M	AC	Pan	U	HM	6/12	3.0	8.1	17.3			Vit		PIE
23	Nathan	33	M	AC	Ant	U	6/18	6/9	1.0	17.3	14.6			Vit	JP	HLA
24	Shanthi	29	F	A	Ant	U	6/24	6/6	0.5	28.0	14.6				VC	HZKU
25	Sujatha	43	F	SA	Ant	U	6/6	6/12	1.0	17.3	12.2			CME		IP
26	Ramuthai	40	F	SA	Pan	U	6/18	1/2/60	1.0	12.2	10.2		+	Vit		EE
27	Ambuli	29	F	SA	Ant	Alt U	6/18	6/60	1.0	17.3	17.3				IDE	HLA
28	Murugan	17	M	AC	Ant	U	6/36	6/6	1.0	15.9	14.6				DU	HZKU
29	Rajan	32	F	AC	Pan	U	6/9	6/24	1.0	17.3	15.9					IP
30	Jeevanandhan	9	M	AC	Pan	U	6/9	CFCF	1.0	14.6	20.4			Vit	LL	LE
31	Ramesh	35	M	REC	Pan	U	6/6	6/36	2.0	14.6	12.2		+	Vit	LBA	HLA

32	Duraisamy	35	M	SA	Pan		6/36	6/6	1.0	17.3	18.9				DL	HZKU
33	Sornam	42	F	Chr	Pan		PL+	6/9	0.5	7.8	17.3	G	+	Vas	OPT	TBA
34	Rajamani	48	M	AC	Ant		6/36	6/18	1.0	20.6	12.2					STE
35	Sundaram	32	M	AC	Pan		6/6	6/24	1.0	14.6	12.2			Vit	JP	HLA
36	Saroja	30	F	AC	Ant		6/9	6/12	1.0	14.6	18.9				VU	HZKU
37	Mani	35	M	REC	Pan		6/18	3/60	0.5	17.3	12.2			Vit	OPT	TBA
38	Chinnasamy	55	M	AC	Ant		6/24	6/60	1.5	12.2	14.6					STE
39	Ranganayaki	58	F	SA	Pan		6/9	6/24	0.5	14.6	12.2			Vit		STE

ABBREVIATIONS

IP - IDIOPATHIC

HLA - HLA B 27

LEP - LEPTOSPIROSIS

PU - PHACOANATHYLACTIC UVEITIS

PU - PHACOTOXIC UVEITIS

STE - STERILE ENDOPHALMITIS

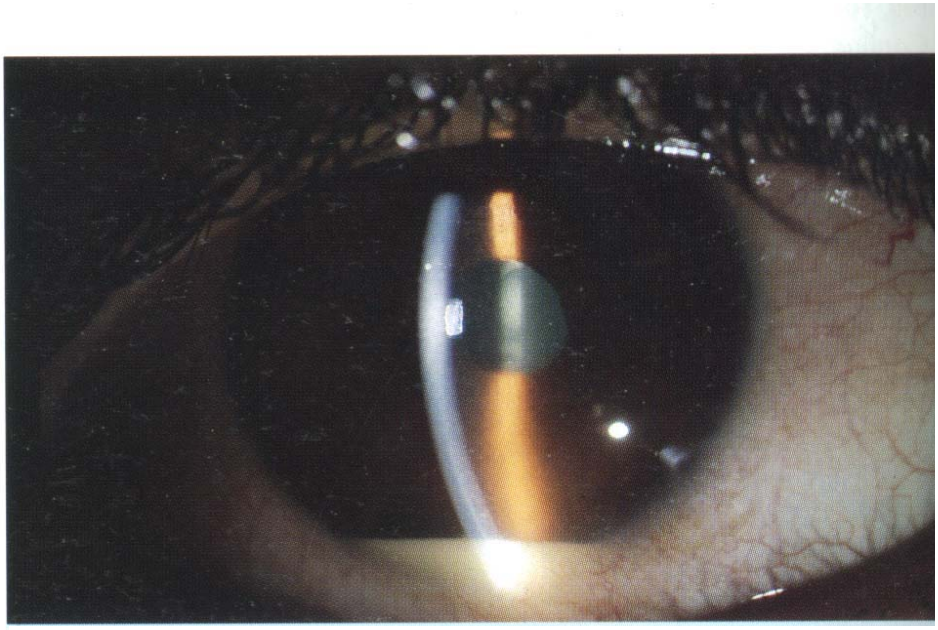
HZKU- HERPES KERATO UVEITIS

LM - LEUKEMIA

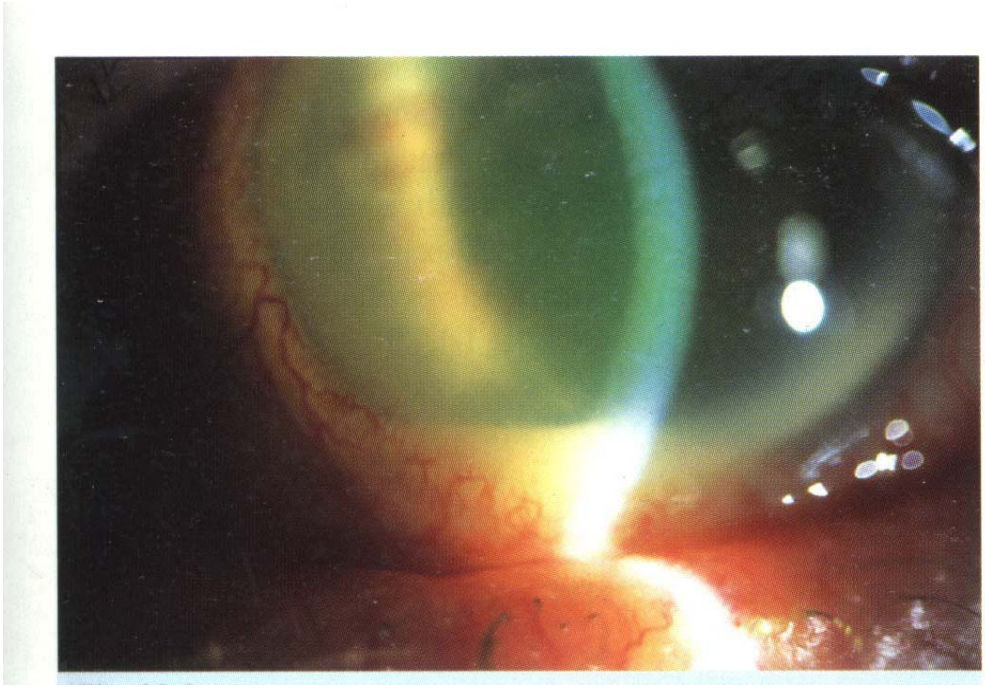
TBA	-	TB ASSOCIATED	JP	-	JOINT PAIN
LBP	-	LOW BACK PAIN	VE	-	VESICULAR ERUPTION
VAS	-	VASCULITIS	PAN	-	PAN UVEITIS
SI	-	SACROILITIS	OPT	-	OLD PULMONARY TUBERCULOSIS
VIT	-	VITRITIS	CME	-	CYSTOID MACULAR EDEMA
DE	-	DISC EDEMA	AC	-	ACUTE
SA	-	SUB ACUTE	CHR	-	CHRONIC
REC	-	RECURRENT	ANT	-	ANTERIOR

PIE - POST OPERATIVE INFECTIOUS ENDOPHTHALMITIS

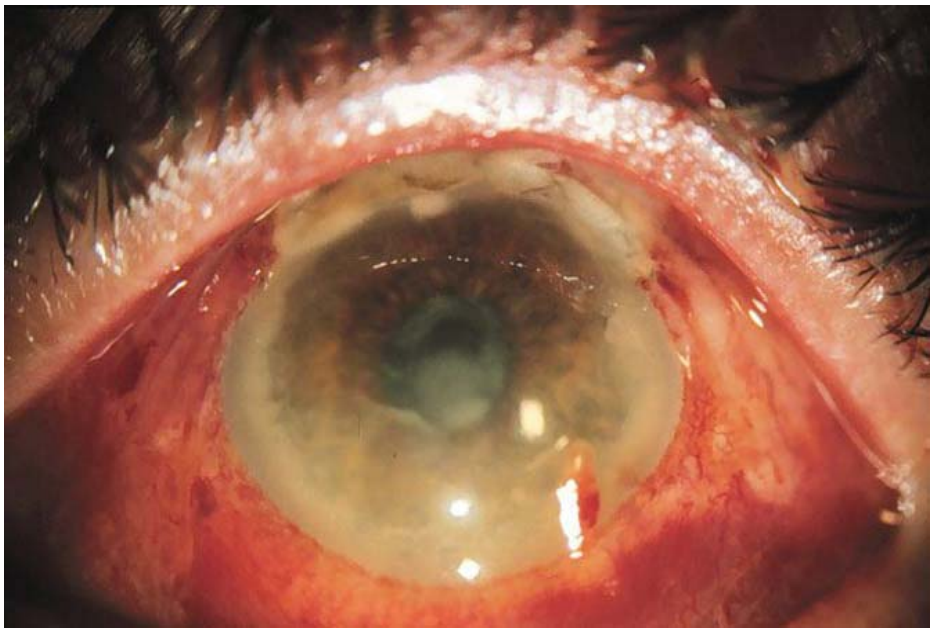
HLA B 27 ASSOCIATED HYPOPYON UVEITIS



HERPETIC KERATO UVEITIC WITH HYPOPYON

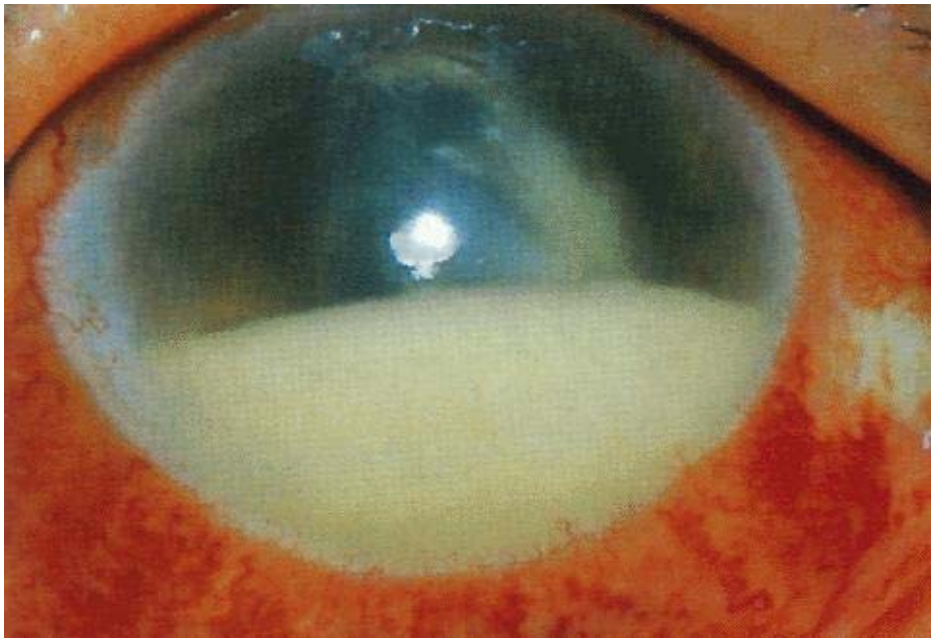


PHACOTOXIC UVEITIS WITH HYPOPYON

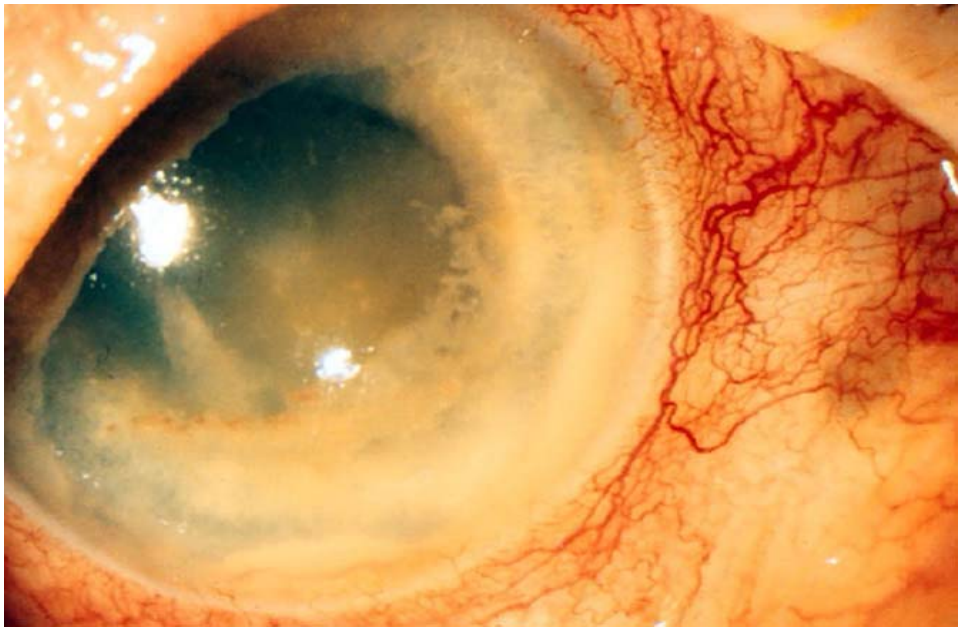


POST OPERATIVE INFECTIONS ENDOPHTHALMITIS WITH HYPOPYON

IDIOPATHIC UVEITIS WITH HYPOPYON



POSTOPERATIVE STERILE ENDOPTHALMITIS WITH HYPOPYON



ANATOMY OF IRIS AND CILIARY BODY